Bandolier 78

What do we think?
What do we know?
What can we prove?

Evidence-based health care **£3.00** **June 2000 Volume 7 Issue 8**

Getting Bandolier in 2001

A reader recently described ***Bandolier*** as an "almost unique source of completely unbiased information". It is independent of the politics of health policy on one side and commercial interests on the other. ***Bandolier*** aims to provide evidence of the research findings in healthcare in a simple, understandable way. Surveys in Wessex [1] and Northern & Yorkshire [2] underline ***Bandolier***'s place in delivering evidence-based information to professionals.

Bandolier maintains its relevance through frequent lectures and discussions about current issues in health care. The feedback we receive is that ***Bandolier*** is an essential resource and much valued.

Where we are now

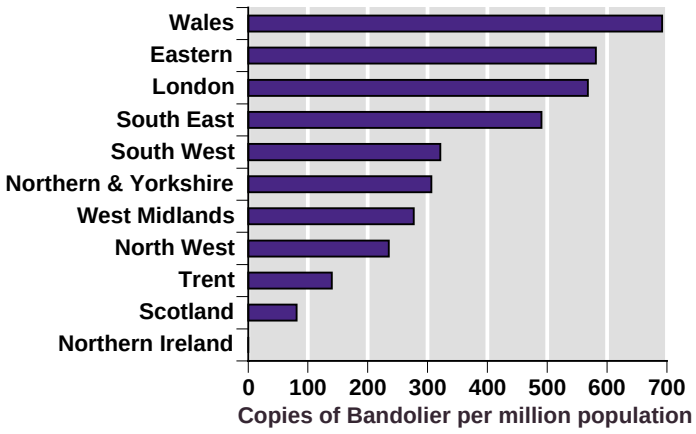
The present distribution arrangements grew like Topsy, and are expensive. Distributing ***Bandolier*** costs as much or more than getting the words on paper. A new system of distribution for PCG/Ts, HAs, Trusts and other healthcare organisations will start from january 2001. It might help tackle the unequal "postcode distribution" (Figure) that has resulted from our Topsy development.

The new arrangements

Bandolier will continue to have central support from NHS R&D, but budgets cannot support the continually expanding circulation. For PCGs, PCTs, Trusts and Health Authorities it is possible to deliver a box of 100 copies of ***Bandolier*** each month for a year for a single payment of £600 (50p per copy). This will reduce the individual cost of an annual subscription from £36 on an individual basis to £5. It will allow

Continued on page 8 col 2

Figure: Distribution of *Bandolier* in the UK



Diagnosing obstructive airway disease

Bandolier wishes it had the EBM equivalent of a Nobel prize to hand out. The prize for the most important contribution to the evidence-base of diagnosis would certainly go to the CARE group (***Bandolier*** 66) investigating the diagnosis of obstructive airway disease [1]. The evidence base in diagnosis and diagnostics is so awful it beggars belief, so when an innovative method using clinicians around the world collaborating through the Internet comes up with the right stuff it is as welcome as a cold glass of water in the desert.

The present study derived from a systematic review of diagnostic criteria for obstructive airways disease (OAD) [2]. It sought physical signs for differentiating between patients with OAD and those with normal pulmonary function. There were many criteria mentioned, but no one sign was found in more than a third of studies. For each of the four most commonly used physical signs the range of diagnostic accuracy from the literature was huge. Positive likelihood ratios spanned the range from about 1 to over 10: from useless to highly predictive.

They also examined the quantity and quality of evidence from systematic reviews for a variety of signs for different conditions. There were few high-quality studies, and those there were were small. The bottom line was that we had little or no objective proof of the quality of diagnostic accuracy of the clinical examination for OAD.

Study

Investigators from around the world were recruited via the CARE Internet site (www.carestudy.com) and the evidence-based email discussion group. Participating groups had at least one physician and one spirometrist and enrolled at least four consecutive patients from each of three categories:

In this issue

1 Patients known to have chronic OAD. This was defined as prior pulmonary function test results less than fifth percentile, patient self report of chronic OAD, bronchitis or emphysema, or use of bronchodilators or steroids.
2 Patients suspected of having OAD who did not fulfil criteria of known OAD but were referred for suspected OAD or the physician thought that OAD was a diagnostic possibility.
3 Patients neither known nor suspected of having OAD.

There were various exclusions, like reversible airway obstruction such as asthma, those with terminal illnesses, and concomitant serious medical conditions.

All the patients underwent clinical examination and independent blinded spirometry. Diagnostic criteria chosen for examination based on the earlier systematic review and consensus were:

- Self reported history of chronic OAD
- Smoking history
- Laryngeal height (distance between the top of the thyroid cartilage and the suprasternal notch: At end-expiration with the patient sitting up, looking straight ahead and with hands relaxed in his/her lap, palpate the top of the thyroid cartilage which is readily identified by the notch on its superior edge. Hook your index finger over the thyroid cartilage and using the rest of your fingers, measure the distance from the top of the thyroid cartilage to the sternal notch in finger-breadths. Convert these finger-breadths to centimetres and record this distance. http://www.carestudy.com/CareStudy/PREOP1/manoeuvre1.asp)
- Laryngeal descent (Maximum laryngeal height was measured at end of inspiration and minimum largyngeal height at end of expiration)
- Wheezing

Each patient underwent a standard protocol for spirometry within 30 minutes of the clinical examination. The gold standard definition of OAD was FEV1 and FEV1-FVC ratio less than the fifth percentile.

Results

Twenty-five investigator groups in 14 countries recruited 322 patients in one month. After excluding some with asthma the final sample size was 309. Likelihood ratios were calculated for all patients, and for those without known chronic OAD. They key indicators were self-reported history of chronic OAD, smoked more than 40 pack years, age 45 or more, maximum laryngeal height 4 cm or less.

For all patients, if all four factors were present the likelihood ratio was a massive 221 (Table). Given a population in which the prior likelihood of OAD was 10%, the post-test probability would be 96%. This would essentially rule in OAD. When all four factors were absent, the post-test probability would be about 1%, essentially ruling out the diagnosis. Where there was no prior history, the three remaining factors achieved much the same result.

Comment

This study triples the number of patients and increases the number of clinicians ten fold over the previous rigorous examination of diagnosis of chronic OAD. It demonstrates that exemplary information about diagnosis can be achieved quickly by use of the Internet. Investigators were involved from Argentina, Australia, Canada, Chile, Colombia, England, Italy, New Zealand, Romania, Spain, Saudi Arabia, United Arab Emirates, and the United States. It is a model for diagnostic testing research, and shows that a number of different issues can quickly be accomplished with a bit of thought. Just imagine what could be done if real resource was put into sorting out diagnostic testing.

This tells us which clinical criteria are important. If spirometry is available, of course it should be used. Where it isn't the data provide useful diagnostic support for the clinician. More please.

Reference:

1 SE Straus, FA McAlister, DL Sackett, JJ Deeks for the CARE-COAD1 Group. The accuracy of patient history, wheezing, and laryngeal measurements in diagnosing obstructive airway disease. JAMA 2000 283: 1853-1857.
2 FA McAlister, SE Straus, DL Sackett. Why we need large, simple studies of the clinical examination: the problem and a proposed solution. Lancet 1999 354: 1721-24.

Table: Likelihood ratios for the four important diagnostic criteriafor chronic obstructive airways disease

	Likelihood ratios			
	All 309 patients		233 patients without known chronic OAD	
Diagnostic element	Factor present	Factor absent	Factor present	Factor absent
Self-reported history of OAD	7.3	0.5		
Smoked more than 40 pack-years	8.3	0.8	11.6	0.9
45 years or more	1.3	0.4	1.4	0.5
Maximum laryngeal height 4 cm or less	2.8	0.8	3.6	0.7
All factors	221	0.13	59	0.32

Better health through better lifestyle

Bandolier has reported previously on the major prospective study of 122,000 nurses in the USA, on homocysteine and colon cancer (***Bandolier*** 60) and walking and heart disease (***Bandolier*** 68). The study has also looked at a number of other links between healthy living and health outcomes.

The big question, though, is whether combing all the different aspects of healthy living makes a substantial difference to health outcomes. Put in a personal way, if ***Bandolier*** were to give up smoking, start drinking, lose weight, eat properly and take some exercise, would it make any difference? The answer seems to be that it would not only make a difference, but reduce the chance of heart attack or stroke by about 80% over 14 years or so [1].

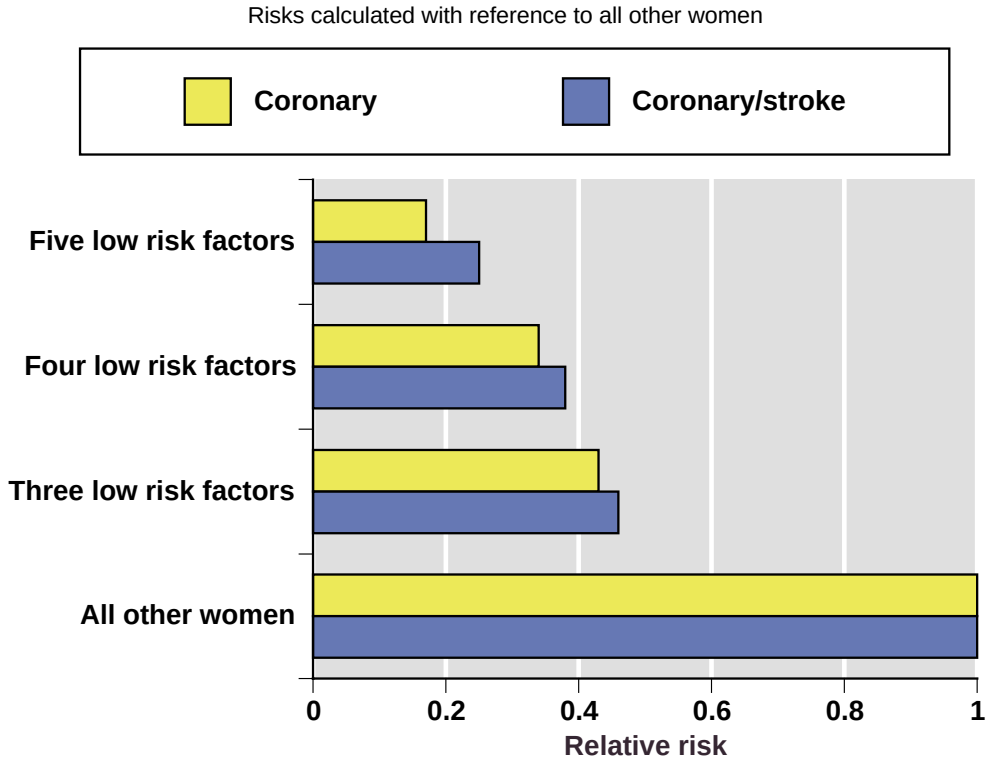
Study

The US Nurses' health study is a cohort of 122,000 female nurses aged 30 to 55 years in 1976, and who have been followed prospectively with bi-annual questionnaires to update information on known or suspected risk factors. This analysis examined the benefits (or otherwise) of lifestyles that combined several features individually known to affect risk of heart disease. Women considered at low risk had the following features:

- Diet: if they scored in the highest 40% of the cohort on a composite measure based on a diet low in trans fat and glycaemic load (extent to which blood sugar may be raised), high in fibre, marine fatty acids, folate and high ratio of polyunsaturated to saturated fat.
- Body mass index: a body mass index (weight in kg divided by the square of the height in metres) of less than 25.
- Physical activity: took 30 minutes of vigorous or moderate activity a day, including brisk walking.
- Alcohol consumption: consumed an average of 5 g of alcohol a day, equivalent to about half a glass of wine.
- Smoking: never smoked or stopped smoking.

Figure and Table: Effect of low risk factor lifestyle on risk of coronary or cardiovascular events (heart attack plus stroke)

Group		Percentage of women	Relative risk (95% CI) Coronary events	Relative risk (95% CI) Coronary events or stroke
Three low risk factors	Good diet Nonsmoking Enough exercise	12.7	0.43 (0.33 to 0.55)	0.46 (0.37 to 0.55)
Four low risk factors	Good diet Nonsmoking Enough exercise Body mass index low	7.2	0.34 (0.23 to 0.52)	0.38 (0.28 to 0.51)
Five low risk factors	Good diet Nonsmoking Enough exercise Body mass index low Enough alcohol	3.1	0.17 (0.07 to 0.41)	0.25 (0.14 to 0.44)



Results

In 14 years of follow up, there were 1128 heart attacks and 705 strokes. The more low risk factors you have in your lifestyle, the lower your risk of heart attack or stroke (Figure and Table). The implications are that in women 82% of heart attacks (95% CI 58% to 93%) and 74% of heart attacks or strokes (55% to 86%) are preventable by having a good lifestyle.

Comment

Only a limited set of variables were considered, but they are variables that any individual can control. We can choose to stop smoking or not smoke. We can choose to have a good diet. We can choose to take sufficient exercise. We can choose to lose weight, or ensure that our weight remains appropriate to our height. We can choose to

drink a moderate amount of alcohol (at least some of us can, as this would be impossible for certain devout religious groups).

The bottom line is that we can choose to put ourselves in the way of heart attacks or strokes, or by behaviour avoid them. The same behaviour is also likely to reduce the risk of cancer, and perhaps other diseases. How to lead a healthy life is something that ***Bandolier*** is asked increasingly. For our own convenience we have drafted a simple 10-point plan for this, and we reproduce it in the box. It is based on studies of high quality carried in ***Bandolier***.

Reference:
1 MJ Stampfer et al. Primary prevention of coronary heart disease in women through diet and lifestyle. New England Journal of Medicine 2000 343: 16-22.

BANDOLIER'S SUMMARY OF ADVICE ON HEALTHY LIVING

This sheet is a quick summary of ten lifestyle tips to help avoid seeing a doctor about heart disease or cancer, based on good quality information. For more details, see the healthy living pages off the ***Bandolier*** home page at www.ebandolier.com.

1 Eat whole grain foods (bread, or rice, or pasta) on four occasions a week. This will reduce the chance of having almost any cancer by 40%. Given that cancer gets about 1 in 3 of us in a lifetime, that's big advice.

2 Don't smoke. If you do smoke, stop. Nicotine patches, gum or inhaler won't help much, and acupuncture won't help at all. Try to reduce your smoking, as there is a profound dose-response (the more you smoke, the more likely you are to have cancer, or heart or respiratory disease). So cut down to below five cigarettes a day and leave long portions of the day without a cigarette.

3 Eat at least five portions of vegetables and fruit a day, and especially tomatoes (including ketchup), red grapes and the like, as well as salad all year. This protects against a whole variety of different nasty things:
- It reduces the risk of stroke dramatically
- It reduces the risk of diabetes considerably
- It will reduce the risk of heart disease and cancer

4 Use Benecol instead of butter or margarine. It really does reduce cholesterol, and reducing cholesterol will reduce the risk of heart attack and stroke even in those whose cholesterol is not particularly high.

5 Drink alcohol regularly. The type of alcohol probably doesn't matter too much, but the equivalent of a couple of glasses of wine a day or a couple of beers is a good thing. The odd day without alcohol won't hurt either. Think of it as medicine.

6 Eat fish. Eating fish once a week won't stop you having a heart attack in itself, but it reduces the likelihood of you dying from it by half.

7 Take a multivitamin tablet every day, but be sure that it is one with at least 200 micrograms of folate. The evidence is that this can substantially reduce chances of heart disease in some individuals, and it has been shown to reduce colon cancer by over 85%. It may also reduce the likelihood of developing dementia. Folate is essential in any woman contemplating pregnancy because it will reduce the chance of some birth defects.

8 If you are pregnant or have high blood pressure, coffee is best minimised. For the rest of us drinking four cups of coffee a day is likely to reduce our chances of getting colon cancer and Parkinson's disease.

9 Get breathless more often. You don't have to go to a gym or be an Olympic marathon runner. Simply walking a mile a day, or taking reasonable exercise three times a week (enough to make you sweat or glow) will substantially reduce the risk of heart disease. If you walk, you don't dawdle. Make it a brisk pace. One of the benefits of regular exercise is that it strengthens bones and keeps them strong. Breaking a hip when elderly is a very serious thing.

10 Check your height and weight on a chart to see if you are overweight for your height. Your body mass index is the weight in kilograms divided by the height in metres squared: for preference it should be below 25. If you are overweight, lose it. This has many benefits. There is no good evidence on simple ways to lose weight that work. Crash diets don't work. Take it one step at a time, do the things that are possible now, and combine some calorie limitation with increased exercise. The good news is that in a few years time we may have some appetite suppressants to make it easier.

Better prescribing of NSAIDs

Providing information to doctors about better prescribing, also called academic detailing, has been shown to be a good thing. A Cochrane review [1] of 18 randomised or quasi randomised trials found positive effects on practice in all trials, but only one measured a patient outcome. A new study from Adelaide [2] demonstrates an apparent remarkable decrease in admissions for perforations, ulcers and gastrointestinal bleeding following detailing about appropriate use of NSAIDs.

Study

The study was conducted in a particular area of metropolitan Adelaide with a population of 154,000. Two surgery visits were made in 1992. These visits focussed on better use of prescribed NSAIDs. **The visits were preceded by a review of the literature, with a written summary of useful information prepared, and externally reviewed by experts and opinion leaders.**

Printed materials providing a source of **"balanced, unbiased information"** were then left at each doctor visit. The programme highlighted the extensive use of NSAIDs and large number of adverse reactions, specifically in high use and high risk groups.

Just under 90% of the 210 doctors practising in the area (80% of whom were GPs) received the service conducted by pharmacists with teaching hospital clinical experience (and most continued to receive advisory visits from the pharmacists about other areas of prescribing). A neighbouring comparison area of 72,000 people did not have the intervention.

Outcomes measured for NSAID use were defined daily doses of NSAIDs prescribed per person per day, and units of NSAIDs delivered to pharmacies from manufacturers and wholesale suppliers. Hospital admissions were monitored for persons with ICD codes indicating ulceration or bleeding events in the upper GI tract with or without bleeding in the intervention and comparison area.

Results

NSAID prescribing in the intervention area fell (Table). Two to four years after the visits prescribing in the intervention area was 9% lower in the intervention area compared to the control area. Two years after the intervention supplies of NSAIDs to pharmacies were 25% lower.

Over the period 1986 to 1998 hospital admission rates for upper GI tract ulceration or bleed in the comparison area was unchanged at about 14 per 100,000 people (Table). In the intervention area rates were rising before the intervention to a peak of about 20 per 100,000 before the intervention. Thereafter rates began to fall, and by 1998 were about 6 per 100,000, a fall of 70% from the peak.

Comment

There is much to say about this. The study could be criticised in a number of ways, but studies of every day interventions are hard to do. The results are remarkable because we are given patient outcomes, starkly unchanged in the comparison area but with big changes in the intervention area. The size and duration of the effect astonish.

But wait. If one thing shines out from this study it is the way in which the intervention was made. Evidence was gathered, it was weighed, it was honed by experts and opinion leaders, and it was "balanced, unbiased information". The doctors who received it were treated as the responsible people they were, and not like mushrooms.

Perhaps this is the secret. If so, this is a clear lead for how PCGs, of about the same average size as the intervention area in this study, could make a difference.

Reference:
1 T O'Brien et al. Educational outreach visits: effects on professional practices and health care outcomes. Cochrane Library 1999 issue 4.
2 FW May et al. Outcomes of an educational-outreach service for community medical practitioners: non-steroidal anti-inflammatory drugs. Medical Journal of Australia 1999 170: 471-474.

Table: Outcomes of NSAID prescribing - use of NSAIDS and hospital admissions for perforation and ulcers

Outcome	Period	Intervention area	Comparison area
NSAID prescribed	94-96 compared with 91	-16%	-7%
NSAID delivered to pharmacies	94 compared with 91	-23%	+5%
Hospital admissions for perforation or ulceration	1992	20/100,000	14/100,000
Hospital admissions for perforation or ulceration	1998	6/100,000	14/100,000

STATINS REDUCE RISK OF FRACTURES

Statins are widely prescribed because they lower cholesterol levels in blood and reduce the risk of cardiovascular mortality and mortality in hyperlipidaemia. Since the early 1990s some of them have also been known to increase bone formation and bone volume in laboratory animals. There is also preliminary evidence that statins increase bone density in humans. We now have three observational studies [1-3] that statins are likely to reduce fractures.

Studies

All three studies had similar case-control architectures. From databases in the UK and USA patients with fractures were matched with controls and the use of statins and other lipid lowering drugs examined. Possible confounding factors were carefully taken into account, and in all studies patients with conditions that could confuse the analyses (like osteoporosis, cancer, and alcoholism) were excluded. Two studies looked at women and men, and one at women only. Two looked at almost all fractures, and one at hip fracture. All the patients were 50 years or older.

Results

The main characteristics and results of the three studies are shown in Table 1. There was a remarkable similarity between the results, with an overall reduction in fractures, and perhaps particularly hip fractures. Current use and amount of statins consumed in the recent past both gave greater statistical significance. There was a use-response relationship for hip fracture in one study [2]. Use of lipid lowering agents other than statins was not associated with reduced risk of fracture in any study.

Comment

It looks as if use of statins reduces the risk of fracture by about half, and perhaps hip fracture by more than half. The crude fracture rate for people taking any lipid lowering drug was half that for people with untreated hyperlipidaemia or matched controls who neither had hyperlipidaemia and never took lipid lowering drugs (Figure) in the UK [1].

There is a biological plausibility for the effect, both because statins are active in the complex biochemistry of bone regulation, and because they have been demonstrated to increase bone formation in animal experiments [4]. But this is not a green light to prescribe statins to prevent fractures. All it does for now is to give us an additional warm glow that the statins we prescribe may be doing more good than we thought, while remembering that an effect seen in observational studies is not always replicated in randomised trials. Not all statins might be the same, and perhaps doses and delivery routes may have to be different to get effects on bone. Right now it is a case of "watch this space".

Reference:

1 CR Meier et al. HMG-CoA reductase inhibitors and the risk of fractures. JAMA 2000 283: 3205-3210.

2 PS Wang et al. HMG-CoA reductase inhibitors and the risk of hip fractures in elderly patients. JAMA 2000 283: 3211-3216.

3 KA Chan et al. Inhibitors of hydroxymethylglutaryl-CoA reductase and risk of fracture among older women. Lancet 2000 355: 2185-2188.

4 SR Cummings, DC Bauer. Do statins prevent both cardiovascular disease and fracture? JAMA 2000 283: 3255-3257.

Figure: Crude fracture risk by treatment [1]

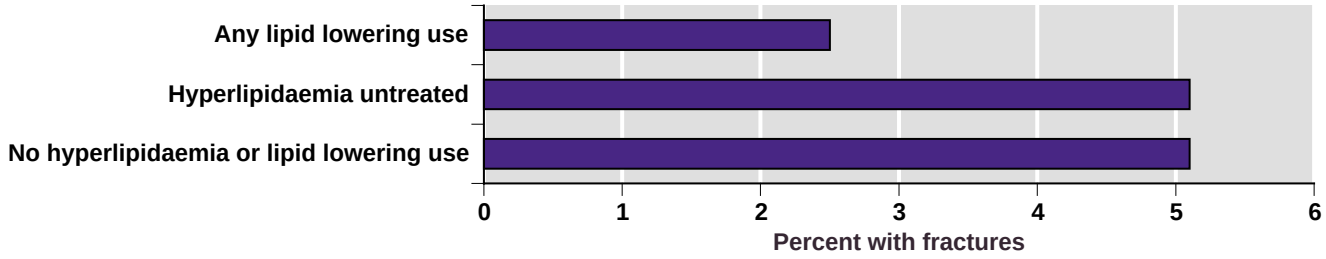


Table: Summary of observational studies

Reference	Base population	Cases	Controls	Main results (odds ratios with 95% CI)
Meier et al [1]	Women and men aged 50-89 years in the UK from GPRD database (late '80s to 1998) taking statins, other lipid lowering drugs, with diagnosis of hyperlipidaemia not on treatment, plus randomly selected patients with none of these indications.	3940 individuals with a first time fracture of femur, humerus, hand, wrist, lower arm, vertebrae, clavicle, foot, or unspecified site	23379 controls without fracture matched on a 1:6 basis with cases	Current use 0.55 (0.44 to 0.69) Fractured femur/current use 0.12 (0.04 to 0.41) Significantly reduced risk for any number of prescriptions, any type of fracture, and recent use, but not past use (no use for 90 days or more). No effect with other lipid lowering drugs
Wang et al [2]	Women and men aged 65 years and older years in New Jersey from Medicare or Medicaid or Pharmacy programs for aged.	1222 cases with hip fracture in 1994 with no prior hip fracture	4888 controls without fracture matched on a 1:4 basis with cases	Current use 0.29 (0.10 to 0.81) Use in past 6 months 0.50 (0.33 to 0.76) Use in past 3 years 0.57 (0.40 to 0.82) Significant use-response, with lower risk with greater statin use over 3 years, with risk for highest use quartile of 0.37 (0.17 to 0.82). No effect with other lipid lowering drugs
Chan et al [3]	Women aged 60 years or older in October 1994 with continuous benefits to 1996 in six HMOs in the USA	928 cases of fracture of hip, humerus, tibia, vertebrae or wrist over one year	2747 matched controls without fracture	For women with more than 13 dispensings of statins there was a statistically reduced rate of 0.48 (0.27 to 0.83). Lower numbers of dispensings were not statistically different. No effect with other lipid lowering drugs

Note that all the results were generated after adjusting for a variety of confounding factors using a number of different methods

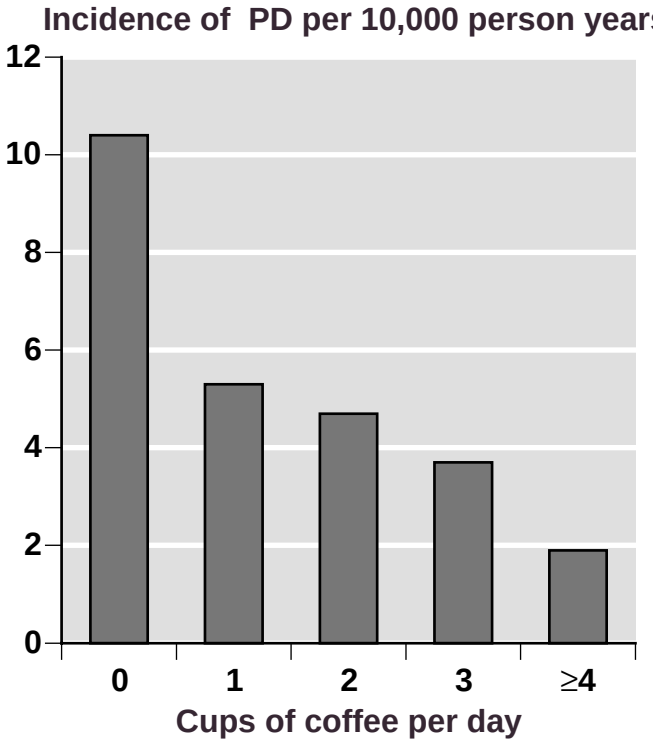
Coffee and Parkinson's disease

How big is your coffee mug? How much do you drink every day? There may be all sorts of reasons for worrying about excess coffee of caffeine intake, but for those, like ***Bandolier***, addicted to double espressos, there is some good news linking higher intake of coffee with a lower incidence of Parkinson's disease [1].

Study

This was part of the Honolulu Heart Programme, a prospective study of 8,000 men of Japanese ancestry aged 45 to 68 years in Oahu in 1965. The survey has continuous surveillance of hospital and death records, with examinations at various times up to 1995.

Figure: Incidence of Parkinson's disease and coffee drinking



At enrolment a detailed nutrient record was performed, verified by a full week dietary record in a subset of men. The record included details about coffee and tea intake (type and quantity) and about other caffeine containing drinks or sources. Other information, like smoking history, was also recorded.

Incident cases of Parkinson's disease were obtained through a full review of all hospital records, ongoing reviews of death certificates, and a cross check with local neurologists. Between 1991 and 1993 all subjects were examined and questioned about any history of Parkinson's disease, including signs and symptoms, and any medicines likely to be taken with someone with Parkinson's disease. A further survey was conducted between 1994 and 1996. All diagnoses were confirmed by a consensus of two neurologists using standard criteria.

Results

Two men had Parkinson's disease at the start of the study, so there was information on 8004 men whose intake was determined in 1965 and who were followed up over 30 years. Their median age at enrolment was 53 years (range 45 to 68 years), and the median duration of follow up was 27 years (0.8 to 30 years). The median age of development of Parkinson's disease in 102 men was 74 years (range 54 to 89 years).

The Table shows the numbers of men in each category of coffee intake (***Bandolier*** has used the number of 8 fluid ounce mugs, each equivalent to two small cups, rather than fluid ounces or millilitres), the crude 30 year incidence, the age adjusted incidence per 10,000 person years, and the adjusted hazard ratio. The same information is available for quintiles of total daily caffeine intake.

Table: Coffee and caffeine consupmtion and Parkinson's disease

Daily coffee consumption (mugs)	Parkinson disease / Total	Percent (95% CI)	Age-adjusted incidence per 10,000 person-years	Adjusted relative hazard ratio (95% CI)
0	32/1286	2.5 (1.6 to 3.3)	10.4	5.1 (1.8 to 14)
1	33/2576	1.3 (0.8 to 1.7)	5.3	2.7 (1.0 to 7.8)
2	24/2149	1.1 (0.7 to 1.6)	4.7	2.5 (0.9 to 7.3)
3	9/1034	0.9 (0.3 to 1.4)	3.7	2.0 (0.6 to 6.4)
≥4	4/959	0.4 (0.0 to 0.8)	1.9	reference
Daily caffeine consumption (mg)	**Parkinson disease / Total**	**Percent (95% CI)**	**Age-adjusted incidence per 10,000 person-years**	**Adjusted relative hazard ratio (95% CI)**
0-123	35/1522	3.3 (1.5 to 3.1)	17.3	3.0 (1.1 to 8.4)
124-208	17/1396	1.2 (0.6 to 1.8)	5.4	1.1 (0.4 to 2.9)
209-287	26/1607	1.6 (1.0 to 2.2)	5.9	1.1 (0.4 to 3.0)
288-420	12/1485	0.8 (0.4 to 1.3)	4.3	0.8 (0.3 to 2.6)
421-2716	6/1481	0.4 (0.1 to 0.7)	5.0	reference

One mug of coffee assumes a volume of 8 fluid ounce or 200 mL;
it is equivalent to two standard cups

Coffee drinkers had a significantly lower incidence of Parkinson's disease than men who did not drink coffee (Table, Figure). The same overall result was apparent for total caffeine intake as for coffee intake (Table). Coffee intake at enrolment was significantly related to Parkinson's disease that occurred during the first and second 15 years of follow up.

Comment

This is the first time, apparently, that this inverse relationship between coffee intake and Parkinson's disease has been demonstrated conclusively. Is there a reason for this? Obviously there must be a reason, and there are many candidates, but right now we don't know what it is. The authors of the paper discuss possible mechanisms in great detail, and it is an interesting read.

Whether the message can be generalised to "Drink coffee and avoid Parkinson's disease" is at least moot. Coffee addicts, though, will take some comfort from this report.

Reference:
1 GW Ross et al. Association of coffee and caffeine intake with the risk of Parkinson disease. JAMA 2000 293: 2674-2679.

THE 2000 INTERNATIONAL WORKSHOP IN EVIDENCE-BASED CLINICAL PRACTICE

11th – 15th September 2000; Oxford, UK.

Chair: Dr Martin Dawes,
NHS R&D Centre for Evidence-Based Medicine
University of Oxford.

This workshop is designed to help clinicial directors (medical, nursing and allied health), senior consultants and medical administrators to develop skills in evidence-based practice, with particular emphasis on formulating clinical questions, finding the evidence and critical appraisal. Facilitators are experienced in both the practice and teaching of EBM.

This Workshop is also part of the Nuffield International Clinical Leadership and Applied Health Informatics Course

More information and application forms may be obtained from:
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http://cebm.jr2.ox.ac.uk/docs/workshopsdoing.html

From Page 1

Table: Cost of Bandolier subscriptions to healthcare organisations in the UK

Number of annual subscriptions	£ per year	£ per subscription
1-9		36.0
10	150	15.0
25	250	10.0
50	400	8.0
75	500	6.7
100	600	6.0
150	800	5.3
200	1000	5.0

each health professional and manager to have his or her own copy, and maximise the knowledge base of the organisation. The costs for different numbers of subscriptions is shown in the Table for the UK (for overseas subscribers the cost will be higher, and will have to reflect shipping charges).

A letter and order form is being posted to all PCGs, PCTs, Health Authorities and Trusts (Chief Executives, PCG leads or Medical Directors) in the UK. Readers in the NHS are likely to belong to one of these organisations, and we would ask those readers to ensure that their organisation is aware that they want to continue receiving ***Bandolier***. There will be some organisations and individuals we miss. A pdf of the form is available from the ***Bandolier*** Internet site, or from Maura Moore (01865 226132, fax 01865 226978).

Intranet possibilities

We also want to explore the possibility of making an electronic form of ***Bandolier*** available for use on Intranets in the future. This would involve downloading the ***Bandolier*** website (including pdf versions of the bulletins) on a monthly or quarterly basis. To cover the work involved we would need to make an annual charge of between £500-1000 for a site license.

References:
1 A McColl, H Smith, P White, J Field. General practitioners' perceptions of the route to evidence based medicine: a questionnaire survey. BMJ 1998 316:361-5.
2 Anon. Access to the evidence base from general practice in Northern and Yorkshire Region. Centre for Reviews and Dissemination. 2000.

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